

AMENDMENTS TO THE CLAIMS:

This listing of claims will replace all prior versions and listings of claims in the application.

Listing of Claims:

1-7. (Cancelled).

8. (Currently amended) ~~The compound according to claim 1~~ A compound,
chosen from

6-(2-butylamino-pyrimidin-4-yl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid (3-pyridin-3-yl-propyl)-amide,

6-(4-hydroxy-3-methoxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid (3-pyridin-3-yl-propyl)-amide,

6-(4-hydroxy-3,5-dimethyl-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid (3-pyridin-3-yl-propyl)-amide,

6-(4-hydroxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid (3-pyridin-3-yl-propyl)-amide,

6-(2-ethylamino-pyrimidin-4-yl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

6-(3-chloro-4-hydroxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

6-(4-hydroxy-3,5-dimethyl-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

4-({[6-(4-hydroxy-3,5-dimethyl-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carbonyl]-amino}-methyl)-benzoic acid,

4-({[6-(4-hydroxy-3-methoxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carbonyl]-amino}-methyl)-benzoic acid,

6-(2-butylamino-pyrimidin-4-yl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid (pyridin-3-yl-methyl)-amide,

6-(3-fluoro-4-hydroxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

6-(4-hydroxy-3-methyl-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

6-[2-(2-morpholin-4-yl-ethylamino)-pyrimidin-4-yl]-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

6-(4-hydroxy-3-methoxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

6-(2-methylamino-pyrimidin-4-yl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

R-3-oxo-6-[2-(1-phenyl-ethylamino)-pyrimidin-4-yl]-2,3-dihydro-pyridazine-4-carboxylic acid (3-phenyl-propyl)-amide,

6-(4-hydroxy-phenyl)-3-oxo-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-benzylamide,

3-oxo-6-pyridin-4-yl-N-[4-(trifluoromethyl)benzyl]-2,3-dihydropyridazine-4-carboxamide,

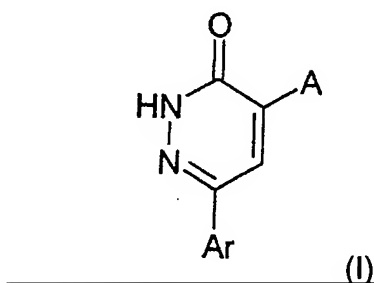
3-oxo-6-pyridin-4-yl-2,3-dihydro-pyridazine-4-carboxylic acid 4-bromo-benzylamide,

3-oxo-6-pyridin-4-yl-N-(pyridin-3-ylmethyl)-2,3-dihydropyridazine-4-carboxamide,

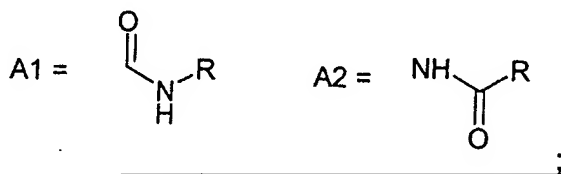
N-(2,4-dichlorobenzyl)-3-oxo-6-pyridin-4-yl-2,3-dihydropyridazine-4-carboxamide,
3-oxo-6-pyridin-4-yl-2,3-dihydro-pyridazine-4-carboxylic acid 4-chloro-2-fluoro-
benzylamide, and

N-(4-chlorobenzyl)-3-oxo-6-pyridin-4-yl-2,3-dihydropyridazine-4-carboxamide;
the racemates, enantiomers, diastereoisomers and mixtures thereof, and the
tautomers or the physiologically acceptable salts thereof.

9. (Currently amended) A method for inhibiting GSK-3 β or the phosphorylation
of the Tau protein in a patient requiring such treatment comprising administering a
physiologically active amount of a compound ~~according to claim 1~~ of formula (I)



wherein A represents A1 or A2



R is unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, aryl,
aryl-(C₁-C₁₀-alkyl)-, heteroaryl, heteroaryl-(C₁-C₁₀-alkyl)-, heterocyclyl,
heterocyclyl-(C₁-C₁₀-alkyl)-, C₃-C₁₀-cycloalkyl, polycycloalkyl,
C₂-C₁₀-alkenyl or C₂-C₁₀-alkynyl,

where the substituents are chosen from halogen, -CN, C₁-C₁₀-alkyl,
-NO₂, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁,

-C(O)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂,
-C(S)NR₁R₂, -NHC(S)R₁, -O-SO₂R₁, -SO₂-O-R₁, oxo, -C(O)R₁,
-C(NH)NH₂, heterocyclyl, C₃-C₁₀-cycloalkyl, aryl-(C₁-C₆-alkyl)-, aryl,
heteroaryl, trifluoromethyl, trifluoromethylsulfanyl and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at
least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen,
trifluoromethyl, trifluoromethoxy or OH;

Ar is unsubstituted or at least monosubstituted aryl or heteroaryl;

where the substituents are chosen from halogen, -CN, NO₂,
C₁-C₁₀-alkyl, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁,
-C(O)NR₁R₂, -NHC(S)R₁, -C(S)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁,
-NHSO₂R₁, -SO₂NR₁R₂, -O-SO₂R₁, -SO₂-O-R₁, aryl, heteroaryl,
aryl-(C₁-C₆-alkyl)-, formyl, trifluoromethyl and trifluoromethoxy,

and the substituents aryl and heteroaryl may further be at least
monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl,
trifluoromethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl,
C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl,
C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl,
where the substituents are chosen from halogen, C₁-C₆-alkyl,
C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl)amino-,
di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl,
trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered, aromatic, mono- or bicyclic heterocycle
containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered, aliphatic, mono- or bicyclic heterocycle containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof;

with the proviso that

(1) A is not -C(O)NH(C₁-C₆-alkyl), when Ar is phenyl which is at least monosubstituted with heterocyclyl or heteroaryl containing nitrogen,

(2) the compound is not 3-{4-(3,4,5-trimethoxyanilincarbonyl)-3-oxo-2,3-dihydropyridazine-6-yl}-2-phenyl-pyrazolo[1,5-a]pyridine; 3-{4-(N-ethoxycarbonylmethyl)-carbamoyl-3-oxo-2,3-dihydro-pyridazine-6-yl}-2-phenyl-pyrazolo[1,5-a]pyridine; 3-{4-(N-carboxymethyl)-carbamoyl-3-oxo-2,3-dihydro-pyridazine-6-yl}-2-phenyl-pyrazolo[1,5-a]pyridine; 6-(4-cyanophenyl)-4[(4-carboxybutyl)-aminocarbonyl]-2H-pyridazin-3-one; or 6-(4-methoxyphenyl)-4-methylcarbamoyl-2H-pyridazin-3-one, and

(3) when A is NHCOCH(CH₃)₂, Ar is not unsubstituted or at least monosubstituted bicyclic heteroaryl.

10. ~~(Currently amended) [[A]] The method for inhibiting GSK-3 β or the phosphorylation of the Tau protein in a patient requiring such treatment comprising administering a physiologically active amount of a compound according to claim 2 according to claim 9, wherein in the formula (I)~~

A is A1;

R is unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, heteroaryl, heteroaryl-(C₁-C₁₀-alkyl)-, heterocyclyl,

heterocyclyl-(C₁-C₁₀-alkyl)-, C₃-C₁₀-cycloalkyl, polycycloalkyl,
C₂-C₁₀-alkenyl or C₂-C₁₀-alkinyl,

where the substituents are chosen from halogen, -CN, C₁-C₁₀-alkyl,
-NO₂, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁,
-C(O)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NH₂SO₂R₁, -SO₂NR₁R₂,
-C(S)NR₁R₂, -NHC(S)R₁, -O-SO₂R₁, -SO₂-O-R₁, oxo, -C(O)R₁,
-C(NH)NH₂, heterocyclyl, C₃-C₁₀-cycloalkyl, aryl-(C₁-C₆-alkyl)-, aryl,
heteroaryl, trifluoromethyl, trifluoromethylsulfanyl and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at
least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen,
trifluoromethyl, trifluoromethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl,
C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl,
C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl,
where the substituents are chosen from halogen, C₁-C₆-alkyl,
C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl)amino-,
di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl,
trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered, aromatic, mono- or bicyclic heterocycle
containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered, aliphatic, mono- or bicyclic heterocycle
containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

11. (Currently amended) ~~[[A]] The method for inhibiting GSK-3 β or the phosphorylation of the Tau protein in a patient requiring such treatment comprising administering a physiologically active amount of a compound according to claim 3 according to claim 9, wherein in the formula (I)~~

R is unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)-, C₃-C₁₀-cycloalkyl, heteroaryl or heteroaryl-(C₁-C₁₀-alkyl)-,

where the substituents are chosen from halogen, -CN, C₁-C₁₀-alkyl, -NO₂, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂, -C(S)NR₁R₂, -NHC(S)R₁, -O-SO₂R₁, -SO₂-O-R₁, oxo, -C(O)R₁, -C(NH)NH₂, heterocyclyl, C₃-C₁₀-cycloalkyl, aryl-(C₁-C₆-alkyl)-, aryl, heteroaryl, trifluoromethyl, trifluoromethylsulfanyl and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoroethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl, C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl, where the substituents are chosen from halogen, C₁-C₆-alkyl, C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl)amino-,

di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl, trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered, aromatic, mono- or bicyclic heterocycle containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered, aliphatic, mono- or bicyclic heterocycle, containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

12. (Currently amended) ~~[[A]] The method for inhibiting GSK-3 β or the phosphorylation of the Tau protein in a patient requiring such treatment comprising administering a physiologically active amount of a compound according to claim 4 according to claim 9, wherein in the formula (I)~~

Ar is unsubstituted or at least monosubstituted phenyl, pyridinyl, pyrimidinyl, pyrazolyl, thiophenyl, isoxazolyl, benzo[b]thiophenyl, benzodioxolyl or thiazolo[3,2-b][1,2,4]-thiazolyl,

where the substituents are chosen from halogen, -CN, NO₂, C₁-C₁₀-alkyl, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -NHC(S)R₁, -C(S)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NH₂SO₂R₁, -SO₂NR₁R₂, -O-SO₂R₁, -SO₂-O-R₁, aryl, heteroaryl, aryl-(C₁-C₆-alkyl)-, formyl, trifluoromethyl and trifluoromethoxy,

and the substituents aryl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoromethoxy or OH;

R1 and R2, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl,
C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl,
C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl,
where the substituents are chosen from halogen, C₁-C₆-alkyl,
C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl) amino-,
di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl,
trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered aromatic, mono- or bicyclic heterocycle,
containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered aliphatic, mono- or bicyclic heterocycle,
containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the
tautomers or the physiologically acceptable salts thereof.

13. (Currently amended) ~~[[A]] The method for inhibiting GSK-3 β or the~~
~~phosphorylation of the Tau protein in a patient requiring such treatment comprising~~
~~administering a physiologically active amount of a compound according to claim 5~~
according to claim 9, wherein in the formula (I)

A is A1;

R is unsubstituted or at least monosubstituted aryl-(C₁-C₆-alkyl)- heteroaryl-
(C₁-C₆-alkyl)- or heterocyclyl-(C₁-C₆-alkyl)-.

where the substituents are chosen from halogen, C₁-C₆-alkyl, -OH, -O-aryl, C₁-C₆-alkoxy, -O-(C₁-C₆-alkyl)-N(C₁-C₆-alkyl)₂, -C(O)OH, -C(O)O-(C₁-C₆-alkyl), -NH₂, -N(C₁-C₆-alkyl)₂, -NH(C₁-C₆-alkyl), -NH(C₁-C₁₀-cycloalkyl), -C(O)NH₂, -C(O)NH-heteroaryl, -C(O)NH-(C₁-C₆-alkyl), -SO₂(C₁-C₆-alkyl), -SO₂NH₂, -C(O)-heterocyclyl, -C(NH)NH₂, heterocyclyl, aryl-(C₁-C₆-alkyl)-, aryl, trifluoromethyl, and trifluoromethoxy.

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₃-alkyl, C₁-C₃-alkoxy, fluorine, chlorine, bromine, trifluoromethyl, trifluoromethoxy or OH;

heteroaryl is imidazolyl, thiophenyl, furanyl, isoxazolyl, pyridinyl, pyrimidinyl, benzoimidazolyl, indolyl or benzodioxolyl;

aryl is phenyl or naphthyl;

heterocyclyl is morpholinyl, piperazinyl or piperidinyl;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

14. (Currently amended) ~~[[A]] The method for inhibiting GSK-3 β or the phosphorylation of the Tau protein in a patient requiring such treatment comprising administering a physiologically active amount of a compound according to claim 6 according to claim 9, wherein in the formula (I)~~

A is A1;

Ar is unsubstituted or at least monosubstituted phenyl, pyridin-4-yl or pyrimidin-4-yl,

where the substituents are chosen from halogen, C₁-C₆-alkyl, -OH, C₁-C₆-alkoxy, -C(O)OH, -C(O)O-(C₁-C₆-alkyl), -NH₂, -N(C₁-C₆-alkyl)₂, -NH(C₁-C₆-alkyl), -NH(C₁-C₁₀-cycloalkyl), -NH(heterocyclyl-(C₁-C₆-alkyl-)), -NH(aryl-(C₁-C₆-alkyl-)), -C(O)NH₂, -C(O)NH-(C₁-C₆-alkyl), aryl, and heteroaryl.

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₃-alkyl, C₁-C₃-alkoxy, fluorine, chlorine, bromine, trifluoromethyl, trifluoromethoxy or OH;

heteroaryl is pyridinyl or pyrimidinyl;

aryl is phenyl or naphthyl;

heterocyclyl is morpholinyl, piperazinyl or piperidinyl;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

15. (Currently amended) ~~[[A]] The method for inhibiting GSK-3 β or the phosphorylation of the Tau protein in a patient requiring such treatment comprising administering a physiologically active amount of a compound according to claim 7~~
according to claim 9, wherein in the formula (I)

A is A1;

R is unsubstituted or at least monosubstituted benzyl, phenylethyl-, phenylpropyl-, pyridinylmethyl-, pyridinylethyl- or pyridinylpropyl-,

where the substituents are chosen from chlorine, bromine, fluorine, trifluoromethyl, and carboxy;

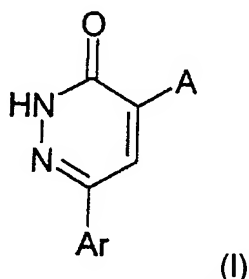
Ar is unsubstituted or at least monosubstituted pyridin-4-yl, pyrimidin-4-yl or phenyl,

where the substituents are chosen from methylamino-, ethylamino-, propylamino-, butylamino-, hydroxy, methoxy, ethoxy, methyl, ethyl, propyl, (phenylethyl)amino-, benzylamino-, and (morpholinylethyl)amino-;

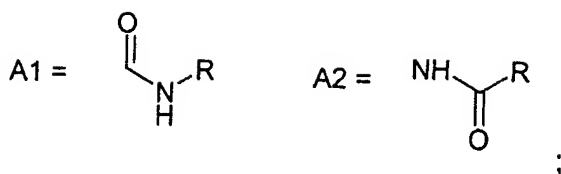
or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

16. (Original) A method for inhibiting GSK-3 β or the phosphorylation of the Tau protein in a patient requiring such treatment comprising administering a physiologically active amount of a compound according to claim 8.

17. (Currently amended) A method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, and immunodeficiency ~~and cancer~~, which method comprises administering a physiologically active amount of a compound ~~according to claim 1~~ of formula (I)



wherein A represents A1 or A2



R is unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, heteroaryl, heteroaryl-(C₁-C₁₀-alkyl)-, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)-, C₃-C₁₀-cycloalkyl, polycycloalkyl, C₂-C₁₀-alkenyl or C₂-C₁₀-alkinyl,

where the substituents are chosen from halogen, -CN, C₁-C₁₀-alkyl, -NO₂, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂, -C(S)NR₁R₂, -NHC(S)R₁, -O-SO₂R₁, -SO₂-O-R₁, oxo, -C(O)R₁, -C(NH)NH₂, heterocyclyl, C₃-C₁₀-cycloalkyl, aryl-(C₁-C₆-alkyl)-, aryl, heteroaryl, trifluoromethyl, trifluoromethylsulfanyl and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoromethoxy or OH;

Ar is unsubstituted or at least monosubstituted aryl or heteroaryl;

where the substituents are chosen from halogen, -CN, NO₂, C₁-C₁₀-alkyl, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -NHC(S)R₁, -C(S)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂, -O-SO₂R₁, -SO₂-O-R₁, aryl, heteroaryl, aryl-(C₁-C₆-alkyl)-, formyl, trifluoromethyl and trifluoromethoxy,

and the substituents aryl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoromethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl,
C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl,
C₂-C₁₀-alkynyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl,
where the substituents are chosen from halogen, C₁-C₆-alkyl,
C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl)amino-,
di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl,
trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered, aromatic, mono- or bicyclic heterocycle
containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered, aliphatic, mono- or bicyclic heterocycle
containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the
tautomers or the physiologically acceptable salts thereof;

with the proviso that

- (1) A is not -C(O)NH(C₁-C₆-alkyl), when Ar is phenyl which is at least
monosubstituted with heterocyclyl or heteroaryl containing nitrogen,
(2) the compound is not 3-{4-(3,4,5-trimethoxyanilinocarbonyl)-3-oxo-2,3-
dihydropyridazine-6-yl}-2-phenyl-pyrazolo[1,5-a]pyridine; 3-{4-(N-
ethoxycarbonylmethyl)-carbamoyl-3-oxo-2,3-dihydro-pyridazine-6-yl}-2-phenyl-
pyrazolo[1,5-a]pyridine; 3-{4-(N-carboxymethyl)-carbamoyl-3-oxo-2,3-dihydro-
pyridazine-6-yl}-2-phenyl-pyrazolo[1,5-a]pyridine; 6-(4-cyanophenyl)-4[(4-
carboxybutyl)-aminocarbonyl]-2H-pyridazin-3-one; or 6-(4-methoxyphenyl)-4-
methylcarbamoyl-2H-pyridazin-3-one, and
(3) when A is NHCOCH(CH₃)₂, Ar is not unsubstituted or at least
monosubstituted bicyclic heteroaryl.

18. (Currently amended) [[A]] The method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, immunodeficiency and cancer, which method comprises administering a physiologically active amount of a compound according to claim 2 according to claim 17, wherein in the formula (I)

A is A1;

R is unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, heteroaryl, heteroaryl-(C₁-C₁₀-alkyl)-, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)-, C₃-C₁₀-cycloalkyl, polycycloalkyl, C₂-C₁₀-alkenyl or C₂-C₁₀-alkinyl,

where the substituents are chosen from halogen, -CN, C₁-C₁₀-alkyl, -NO₂, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂, -C(S)NR₁R₂, -NHC(S)R₁, -O-SO₂R₁, -SO₂-O-R₁, oxo, -C(O)R₁, -C(NH)NH₂, heterocyclyl, C₃-C₁₀-cycloalkyl, aryl-(C₁-C₆-alkyl)-, aryl, heteroaryl, trifluoromethyl, trifluoromethylsulfanyl and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoromethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl, C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl, where the substituents are chosen from halogen, C₁-C₆-alkyl, C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl)amino-, di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl, trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered, aromatic, mono- or bicyclic heterocycle containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered, aliphatic, mono- or bicyclic heterocycle containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

19. (Currently amended) ~~[[A]] The method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, immunodeficiency and cancer, which method comprises administering a physiologically active amount of a compound according to claim 3 according to claim 17, wherein in the formula (I)~~

R is unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)-, C₃-C₁₀-cycloalkyl, heteroaryl or heteroaryl-(C₁-C₁₀-alkyl)-,

where the substituents are chosen from halogen, -CN, C₁-C₁₀-alkyl, -NO₂, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂, -C(S)NR₁R₂, -NHC(S)R₁, -O-SO₂R₁, -SO₂-O-R₁, oxo, -C(O)R₁, -C(NH)NH₂, heterocyclyl, C₃-C₁₀-cycloalkyl, aryl-(C₁-C₆-alkyl)-, aryl, heteroaryl, trifluoromethyl, trifluoromethylsulfanyl and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoroethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl, C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl, where the substituents are chosen from halogen, C₁-C₆-alkyl, C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl)amino-, di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl, trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered, aromatic, mono- or bicyclic heterocycle containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered, aliphatic, mono- or bicyclic heterocycle, containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

20. (Currently amended) [[A]] The method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, immunodeficiency and cancer, which method comprises administering a physiologically active amount of a compound according to claim 4 according to claim 17, wherein in the formula (I)

Ar is unsubstituted or at least monosubstituted phenyl, pyridinyl, pyrimidinyl, pyrazolyl, thiophenyl, isoxazolyl, benzo[b]thiophenyl, benzodioxolyl or thiazolo[3,2-b][1,2,4]-thiazolyl,

where the substituents are chosen from halogen, -CN, NO₂, C₁-C₁₀-alkyl, -OR₁, -C(O)OR₁, -O-C(O)R₁, -NR₁R₂, -NHC(O)R₁, -C(O)NR₁R₂, -NHC(S)R₁, -C(S)NR₁R₂, -SR₁, -S(O)R₁, -SO₂R₁, -NHSO₂R₁, -SO₂NR₁R₂, -O-SO₂R₁, -SO₂-O-R₁, aryl, heteroaryl, aryl-(C₁-C₆-alkyl)-, formyl, trifluoromethyl and trifluoromethoxy,

and the substituents aryl and heteroaryl may further be at least monosubstituted with C₁-C₆-alkyl, C₁-C₆-alkoxy, halogen, trifluoromethyl, trifluoromethoxy or OH;

R₁ and R₂, independently from each other, are

hydrogen;

unsubstituted or at least monosubstituted C₁-C₁₀-alkyl, C₃-C₁₀-cycloalkyl, aryl, aryl-(C₁-C₁₀-alkyl)-, C₂-C₁₀-alkenyl, C₂-C₁₀-alkinyl, heterocyclyl, heterocyclyl-(C₁-C₁₀-alkyl)- or heteroaryl, where the substituents are chosen from halogen, C₁-C₆-alkyl, C₁-C₆-alkoxy, CN, NO₂, NH₂, (C₁-C₆-alkyl) amino-,

di(C₁-C₆-alkyl)amino-, OH, COOH, -COO-(C₁-C₆-alkyl), -CONH₂, formyl, trifluoromethyl and trifluoromethoxy;

heteroaryl is a 5 to 10-membered aromatic, mono- or bicyclic heterocycle, containing one or more heteroatoms chosen from N, O and S;

aryl is phenyl, indanyl, indenyl or naphthyl;

heterocyclyl is a 5 to 10-membered aliphatic, mono- or bicyclic heterocycle, containing one or more heteroatoms chosen from N, O and S;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

21. (Currently amended) ~~[[A]] The method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, immunodeficiency and cancer, which method comprises administering a physiologically active amount of a compound according to claim 5 according to claim 17, wherein in the formula (I)~~

A is A1;

R is unsubstituted or at least monosubstituted aryl-(C₁-C₆-alkyl)- heteroaryl-(C₁-C₆-alkyl)- or heterocyclyl-(C₁-C₆-alkyl)-,

where the substituents are chosen from halogen, C₁-C₆-alkyl, -OH, -O-aryl, C₁-C₆-alkoxy, -O-(C₁-C₆-alkyl)-N(C₁-C₆-alkyl)₂, -C(O)OH, -C(O)O-(C₁-C₆-alkyl), -NH₂, -N(C₁-C₆-alkyl)₂, -NH(C₁-C₆-alkyl), -NH(C₁-C₁₀-cycloalkyl), -C(O)NH₂, -C(O)NH-heteroaryl, -C(O)NH-(C₁-C₆-alkyl), -SO₂(C₁-C₆-alkyl), -SO₂NH₂, -C(O)-heterocyclyl, -

C(NH)NH₂, heterocyclyl, aryl-(C₁-C₆-alkyl)-, aryl, trifluoromethyl, and trifluoromethoxy,

and the substituents aryl, heterocyclyl and heteroaryl may further be at least monosubstituted with C₁-C₃-alkyl, C₁-C₃-alkoxy, fluorine, chlorine, bromine, trifluoromethyl, trifluoromethoxy or OH;

heteroaryl is imidazolyl, thiophenyl, furanyl, isoxazolyl, pyridinyl, pyrimidinyl, benzoimidazolyl, indolyl or benzodioxolyl;

aryl is phenyl or naphthyl;

heterocyclyl is morpholinyl, piperazinyl or piperidinyl;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

22. (Currently amended) ~~[[A]] The method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, immunodeficiency and cancer, which method comprises administering a physiologically active amount of a compound according to claim 6~~ according to claim 17, wherein in the formula (I)

A is A1;

Ar is unsubstituted or at least monosubstituted phenyl, pyridin-4-yl or pyrimidin-4-yl,

where the substituents are chosen from halogen, C₁-C₆-alkyl, -OH, C₁-C₆-alkoxy, -C(O)OH, -C(O)O-(C₁-C₆-alkyl), -NH₂, -N(C₁-C₆-alkyl)₂,

-NH(C₁-C₆-alkyl), -NH(C₁-C₁₀-cycloalkyl), -NH(heterocyclyl-(C₁-C₆-alkyl-)),
-NH(aryl-(C₁-C₆-alkyl-)), -C(O)NH₂, -C(O)NH-(C₁-C₆-alkyl), aryl, and
heteroaryl,

and the substituents aryl, heterocyclyl and heteroaryl may further be at
least monosubstituted with C₁-C₃-alkyl, C₁-C₃-alkoxy, fluorine, chlorine,
bromine, trifluoromethyl, trifluoromethoxy or OH;

heteroaryl is pyridinyl or pyrimidinyl;

aryl is phenyl or naphthyl;

heterocyclyl is morpholinyl, piperazinyl or piperidinyl;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the
tautomers or the physiologically acceptable salts thereof.

23. (Currently amended) ~~[[A]] The method for treating a patient suffering from
a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas
and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential
hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome,
syndrome X, immunodeficiency and cancer, which method comprises administering a
physiologically active amount of a compound according to claim 7 according to claim
17, wherein in the formula (I)~~

A is A1;

R is unsubstituted or at least monosubstituted benzyl, phenylethyl-,
phenylpropyl-, pyridinylmethyl-, pyridinylethyl- or pyridinylpropyl-

where the substituents are chosen from chlorine, bromine, fluorine, trifluoromethyl, and carboxy;

Ar is unsubstituted or at least monosubstituted pyridin-4-yl, pyrimidin-4-yl or phenyl,

where the substituents are chosen from methylamino-, ethylamino-, propylamino-, butylamino-, hydroxy, methoxy, ethoxy, methyl, ethyl, propyl, (phenylethyl)amino-, benzylamino-, and (morpholinylethyl)amino-;

or the racemates, enantiomers, diastereoisomers and mixtures thereof, the tautomers or the physiologically acceptable salts thereof.

24. (Currently amended) A method for treating a patient suffering from a disease chosen from neurodegenerative diseases, strokes, cranial and spinal traumas and peripheral neuropathies, obesity, metabolic diseases, type II diabetes, essential hypertension, atherosclerotic cardiovascular diseases, polycystic ovary syndrome, syndrome X, and immunodeficiency ~~and cancer~~, which method comprises administering a physiologically active amount of a compound according to claim 8.

25. (Original) The method according to claim 17, wherein the neurodegenerative disease is chosen from Alzheimer's disease, Parkinson's disease, frontoparietal dementia, corticobasal degeneration and Pick's disease.

26. (Original) The method according to claim 17, wherein the disease is chosen from type-II-diabetes and Alzheimer's disease.

27-29. (Cancelled).

30. (New) The method according to claim 24, wherein the neurodegenerative disease is chosen from Alzheimer's disease, Parkinson's disease, frontoparietal dementia, corticobasal degeneration and Pick's disease.

31. (New) The method according to claim 24, wherein the disease is chosen from type-II-diabetes and Alzheimer's disease.